

FILE 'USPATFULL' ENTERED AT 22:27:13 ON 15 JUN 2003

L1 2286 S RERADIAT? OR RE-RADIATI?
L2 3 S L1 AND SUNSCREEN?
L3 3 S L1 AND (SUNSCREEN? OR SUNBLOCK?)
L4 25 S L1 AND COSMETIC
L5 0 S L1 (3S) (HARMFUL RO HARM? OR DAMAG?) (3S) SKIN
L6 27 S L1 (3S) SKIN

FILE 'CAPLUS, MEDLINE, EMBASE, SCISEARCH' ENTERED AT 22:36:44 ON 15 JUN 2003

L7 382 FILE CAPLUS
L8 23 FILE MEDLINE
L9 30 FILE EMBASE
L10 254 FILE SCISEARCH

TOTAL FOR ALL FILES

L11 689 S L1
L12 2 FILE CAPLUS
L13 1 FILE MEDLINE
L14 2 FILE EMBASE
L15 4 FILE SCISEARCH

TOTAL FOR ALL FILES

L16 9 S L11 AND (COSMETIC OR SUNSCREEN OR SUNBLOCK OR SKIN)
L17 323 FILE CAPLUS
L18 9 FILE MEDLINE
L19 20 FILE EMBASE
L20 205 FILE SCISEARCH

TOTAL FOR ALL FILES

L21 557 S RE-READIAT? OR RERADIAT?
L22 417 FILE CAPLUS
L23 25 FILE MEDLINE
L24 34 FILE EMBASE
L25 284 FILE SCISEARCH

TOTAL FOR ALL FILES

L26 760 S RE-RADIAT? OR RERADIAT?
L27 2 FILE CAPLUS
L28 1 FILE MEDLINE
L29 2 FILE EMBASE
L30 4 FILE SCISEARCH

TOTAL FOR ALL FILES

L31 9 S L26 AND (COSMETIC OR SUNSCREEN OR SUNBLOCK OR SKIN)
L32 160390 FILE CAPLUS
L33 11327 FILE MEDLINE
L34 14060 FILE EMBASE
L35 59592 FILE SCISEARCH

TOTAL FOR ALL FILES

L36 245369 S EMIT? OR EMSSION
L37 8 FILE CAPLUS
L38 5 FILE MEDLINE
L39 9 FILE EMBASE
L40 10 FILE SCISEARCH

TOTAL FOR ALL FILES

L41 32 S L36 AND (SUNSCREEN OR SUNBLOCK) AND SKIN

FILE 'USPATFULL' ENTERED AT 22:27:13 ON 15 JUN 2003

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L2 3 S L1 AND SUNSCREEN?
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L31 9 S L26 AND (COSMETIC OR SUNSCREEN OR SUNBLOCK OR SKIN)
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L36 245369 S EMIT? OR EMSSION
L37 8 FILE CAPLUS
L38 5 FILE MEDLINE
L39 9 FILE EMBASE
L40 10 FILE SCISEARCH
TOTAL FOR ALL FILES
L41 32 S L36 AND (SUNSCREEN OR SUNBLOCK) AND SKIN

L41 ANSWER 29 OF 32 SCISEARCH COPYRIGHT 2003 THOMSON ISI
 AN 96:452304 SCISEARCH
 GA The Genuine Article (R) Number: UQ162
 TI ULTRAVIOLET SPECTRAL ENERGY DIFFERENCES AFFECT THE ABILITY OF
SUNSCREEN LOTIONS TO PREVENT ULTRAVIOLET-RADIATION-INDUCED
 IMMUNOSUPPRESSION
 AU ROBERTS L K (Reprint); BEASLEY D G; LEARN D B; GIDDENS L D; BEARD J;
 STANFIELD J W
 CS SCHERING PLOUGH CORP, HEALTHCARE PROD, ADV PROD RES, AR-3-59, 3030 JACKSON
 AVE, MEMPHIS, TN, 38151 (Reprint); SCHERING PLOUGH CORP, HEALTHCARE PROD,
 SOLAR RES LABS, MEMPHIS, TN, 38151
 CYA USA
 SO PHOTOCHEMISTRY AND PHOTOBIOLOGY, (JUN 1996) Vol. 63, No. 6, pp. 874-884.
 ISSN: 0031-8655.
 DT Article; Journal
 FS LIFE
 LA ENGLISH
 REC Reference Count: 48
 AB Acute exposure to UV radiation causes immunosuppression of contact
 hypersensitivity (CH) responses. Past studies conducted with unfiltered
 sunlamps **emitting** nonsolar spectrum UV power (wavelengths below
 295 nm) or using excessive UV doses have suggested **sunscreens**
 may not prevent UV-induced immunosuppression in mice. This study was thus
 designed to evaluate critically the effects of different UV energy spectra
 on the immune protection capacity of **sunscreen** lotions. Minimum
 immune suppression doses (MISD), i.e. the lowest UV dose to cause similar
 to 50% suppression of the CH response to dinitrofluorobenzene in C3H mice,
 were established for three artificial UV sources. The MISD for each UV
 source was 0.25 kJ/m(2) for unfiltered FS20 sunlamps (FS), 0.90 kJ/m(2)
 for Kodacel-filtered FS20 sunlamps (KFS), which do not **emit** UV
 power at wavelengths <290 nm, and 1.35 kJ/m(2) for a 1000 W filtered xenon
 arc lamp solar simulator. Using MISD as baseline, **sunscreens**
 with labeled sun protection factors (SPF) of 4, 8, 15 and 30 were tested
 with each UV source to establish their relative immune protection factors.
 The immune protection factor of each **sunscreen** exceeded its
 labeled SPF in tests conducted with the solar simulator, which has a UV
 power spectrum (295-400 nm) similar to that of sunlight. Conversely,
sunscreen immune protection factors were significantly less than
 the labeled SPF in tests conducted with FS and KFS. Comparison of the
 immunosuppression effectiveness spectra showed that relatively small
 amounts of nonsolar spectrum UV energy, i.e. UVC (200-290 nm) and/or
 shorter wavelength UVB (between 290 and 295 nm), produced by FS and KFS
 contributes significantly to the induction of immunosuppression. For
 example, 36.3% and 3.5% of the total immunosuppressive UV energy from FS
 and KFS, respectively, lies below 295 nm. **Sunscreens** absorption
 spectra showed that transmission of immunosuppressive UV energy below 295
 nm for FS was at least eight-fold higher than that for KFS. Compared to
 the solar simulator UV spectrum the transmission of non-solar
 immunosuppressive UV energy through **sunscreens** was >15-fold
 higher for FS and greater than or equal to 1.5-fold higher for KFS. These
 data demonstrate that relevant evaluations of **sunscreens** immune
 protection can only be obtained when tests are conducted with UV sources
 that produce UV power spectra similar to that of sunlight and UV doses are
 employed that are based on established MISD.
 CC BIOPHYSICS; BIOCHEMISTRY & MOLECULAR BIOLOGY
 STP KeyWords Plus (R): CONTACT HYPERSENSITIVITY; HUMAN-**SKIN**; INDUCED
 SUPPRESSION; AMINO BENZOIC ACID; CELL-ACTIVITY; MICE; INDUCTION; DNA;
 PROTECTION; RESPONSES
 RF 94-0898 001; STRATOSPHERIC OZONE DEPLETION; PINATUBO AEROSOL;
 ULTRAVIOLET-B RADIATION; HETEROGENEOUS CHEMISTRY
 RE

L41 ANSWER 27 OF 32 SCISEARCH COPYRIGHT 2003 THOMSON ISI
 AN 97:84982 SCISEARCH
 GA The Genuine Article (R) Number: WD125
 TI Broad-spectrum **sunscreens** with UVA I and UVA II absorbers
 provide increased protection against solar-simulating radiation-induced
 dermal damage in hairless mice
 AU Kligman L H (Reprint); Agin P P; Sayre R M
 CS UNIV PENN, SCH MED, DEPT DERMATOL, PHILADELPHIA, PA 19104 (Reprint);
 SCHERING PLOUGH CORP, HLTH CARE PROD, RES & DEV, MEMPHIS, TN 38151; RAPID
 CYA PRECIS TESTING LAB, CORDOVA, TN
 SO USA
 JOURNAL OF THE SOCIETY OF COSMETIC CHEMISTS, (MAY-JUN 1996) Vol. 47, No.
 3, pp. 129-155.
 Publisher: SOC COSMETIC CHEMISTS, 120 WALL STREET, SUITE 2400, NEW YORK,
 NY 10005-4088.
 ISSN: 0037-9832.
 DT Article; Journal
 LA English
 REC Reference Count: 23
 AB Previous experiments designed to examine **sunscreen** protection
 against chronic UV radiation-induced **skin** damage in hairless
 mice have used radiation sources **emitting** mainly UVB or UVA
 radiation. Because humans are exposed to full-spectrum solar radiation, we
 were interested in examining the efficacy of three **sunscreens**,
 with increasing spectral absorption into the UVA range, against chronic
 solar-simulating radiation (SSR). Three groups of hairless mice received a
 cumulative SSR dose of 10 and 16 times a previously determined minimal
 photoaging dose (MPD) over periods of 18 and 30 weeks. Each twice-weekly
 exposure was designed to equal the SPF value of the first
sunscreen, an SPF-7 **sunscreen** containing the UVB
 absorber octyl methoxycinnamate. The second **sunscreen**, in
 addition to the UVB absorber, contained a UVA II absorber (oxybenzone) and
 had an SPF of 16. The third, with an SPF of 18, contained the UVB and UVA
 II absorbers plus a UVA I absorber (avobenzone). These conditions allowed
 assessment of the effects of UVB and UVA radiation that are normally
 transmitted through all **sunscreens**. Although none of the
sunscreen-treated mice developed erythema, considerable dermal
 matrix damage occurred in the SPF-7 group, with greater damage at 16 MPD
 than at 10 MPD. The SPF-16 **sunscreen** allowed less but clearly
 recognizable damage at both dose points. The SPF-18 **sunscreen**
 with the broadest spectral absorption provided the greatest protection.
 These results support the need for high-SPF broad-spectrum
sunscreen protection that includes the entire UVA spectrum to
 reduce photodamage that results from chronic exposure to sunlight.
 CC CHEMISTRY, APPLIED; DERMATOLOGY & VENEREAL DISEASES
 STP KeyWords Plus (R): MOUSE **SKIN**; ULTRAVIOLET; IRRADIATION;
 RE COLLAGENASE; INVIVO

L41 ANSWER 26 OF 32 SCISEARCH COPYRIGHT 2003 THOMSON ISI
 AN 97:460481 SCISEARCH
 GA The Genuine Article (R) Number: XE030
 TI Oxidative stress and in vivo chemiluminescence in mouse **skin**
 exposed to UVA radiation
 AU Evelson P (Reprint); Ordonez C P; Llesuy S; Boveris A
 CS UNIV BUENOS AIRES, FAC FARM & BIOQUIM, CATEDRA FISICOQUIM, JUNIN 956,
 RA-1113 BUENOS AIRES, DF, ARGENTINA (Reprint); UNIV BUENOS AIRES, FAC FARM
 & BIOQUIM, CATEDRA QUIM GEN & INORGAN, RA-1113 BUENOS AIRES, DF, ARGENTINA
 CYA ARGENTINA
 SO JOURNAL OF PHOTOCHEMISTRY AND PHOTOBIOLOGY B-BIOLOGY, (APR 1997) Vol. 38,
 No. 2-3, pp. 215-219.
 Publisher: ELSEVIER SCIENCE SA LAUSANNE, PO BOX 564, 1001 LAUSANNE 1,
 SWITZERLAND.
 ISSN: 1011-1344.
 DT Article; Journal
 FS LIFE
 LA English
 REC Reference Count: 27
 AB Mouse **skin** was exposed to UVA radiation (320-400 nm). The in
 vivo chemiluminescence of the **skin** was measured after
 irradiation. Chemiluminescence showed a maximum 13-fold increase (control
 emission, 10+/-1 cps cm(-2)) after 45-60 min of exposure to UVA, with no
 further increase with 60 min additional exposure. Spectral analysis of the
emitted chemiluminescence showed that the principal species
emitted in the 400-500 nm range. Topical application with
 alpha-tocopherol (10% v/w) and beta-carotene (1 mM) greatly reduced the
 UVA-induced **skin** chemiluminescence. Thiobarbituric acid reactive
 substance (TEARS) levels were increased by 130% in **skin**
 homogenates after 2 h of exposure to UVA (control value, 77 +/- 14 nmol
 malonaldehyde equivalents (g tissue) (-1)). The activities of antioxidant
 enzymes in **skin** homogenates were decreased after 2 h of
 irradiation: the superoxide dismutase (SOD) activity (control value,
 181+/-10 U SOD (g tissue) (-1)) was decreased by 40% and the catalase
 activity (control value, 1.34+/-0.14 pmol (g tissue) (-1)) was decreased by
 45%. In vivo chemiluminescence appears to be a suitable method for
 following the kinetics of the **skin** oxidative stress processes
 and for testing the effect of topical application with antioxidants and
 photoprotective agents. (C) 1997 Elsevier Science S.A.
 CC BIOPHYSICS; BIOCHEMISTRY & MOLECULAR BIOLOGY
 ST Author Keywords: chemiluminescence; lipid peroxidation; oxidative stress;
 photoprotectors; UVA
 STP KeyWords Plus (R): LIPID-PEROXIDATION; CHEMI-LUMINESCENCE;
 ULTRAVIOLET-RADIATION; MECHANISMS; LIGHT; REPERFUSION; **SUNSCREENS**
 ; EPIDERMIS; MODEL; LIVER

RE

Referenced Author	Year	VOL	PG	Referenced Work
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L41 ANSWER 25 OF 32 SCISEARCH COPYRIGHT 2003 THOMSON ISI
 AN 97:507680 SCISEARCH
 GA The Genuine Article (R) Number: XH200
 TI **Sunscreens** prevent local and systemic immunosuppression of
 contact hypersensitivity in mice exposed to solar-simulated ultraviolet
 radiation
 AU Roberts L K (Reprint); Beasley D G
 CS SCHERING PLOUGH CORP, HEALTHCARE PROD, ADV PROD RES, AR-3-59, 3030 JACKSON
 AVE, MEMPHIS, TN 38151 (Reprint)
 CYA USA
 SO JOURNAL OF PHOTOCHEMISTRY AND PHOTOBIOLOGY B-BIOLOGY, (JUN 1997) Vol. 39,
 No. 2, pp. 121-129.
 Publisher: ELSEVIER SCIENCE SA LAUSANNE, PO BOX 564, 1001 LAUSANNE,
 SWITZERLAND.
 ISSN: 1011-1344.
 DT Article; Journal
 FS LIFE
 LA English
 REC Reference Count: 46
 AB Ultraviolet (UV) irradiation causes the immunosuppression of contact
 hypersensitivity (CH) responses in animals and humans. There are
 conflicting reports regarding the effectiveness of **sunscreens** in
 preventing UV-induced suppression of both local-type CH (induced by the
 application of the contact sensitizer directly to UV-exposed **skin**
) and systemic-type CH (induced by the application of the contact
 sensitizer to an unirradiated **skin** site 3 days after UV
 exposure). The purposes of this study were as follows:
 1. to derive solar simulator UV dose-response curves for the induction
 of local and systemic CH suppression in C3H mice; 2, to establish minimum
 immune suppression doses (MISDs) for local and systemic CH;
 3. to determine the local and systemic immune protection capacity of
 two commercial **sunscreen** lotions with labeled sun protection
 factors (SPFs) of 4 and 8,
 Dose-response curves for the induction of local and systemic CH
 suppression were derived by exposing groups of mice to a range of
 full-spectrum UV doses (0.37-21.4 kJ m⁻²) on two consecutive days
 delivered from a filtered 1000 W xenon arc lamp solar simulator. The
 MISDs, defined as the lowest dose tested to cause approximately 50%
 suppression of the normal CH response, were obtained from the
 dose-response curves. Although the local and systemic immunosuppression
 dose-response curves were not statistically different, the MISD for local
 suppression of CH (1.35 kJ m⁻²) was about fivefold lower than that for
 systemic CH suppression (6.76 kJ m⁻²). The MISD was used as the endpoint to
 determine **sunscreen** immune protection levels. Both
sunscreens, applied at 2 mg cm⁻², provided immune protection
 against the induction of local and systemic CH suppression in mice exposed
 to an effective UV dose of 1 MISD given through the **sunscreen**,
 i.e. 4 MISD to SPF 4 **sunscreen**-protected mice and 8 MISD to SPF
 8 **sunscreen**-protected mice mounted CH responses that were
 significantly greater than those elicited in unprotected mice exposed to 1
 MISD of solar simulated UV radiation. The calculated immune protection
 factors for these **sunscreens** exceeded the level of protection
 predicted by their labeled SPFs, i.e. the local immune protection
 factor of both **sunscreens** was 15 and the systemic immune protection
 factors were 8 for the SPF 4 **sunscreen** and 15 for the SPF 8
sunscreen. Our data show that these two **sunscreens**
 provide levels of immune protection which exceed the levels predicted by
 their labeled SPFs in immunoprotection tests conducted in mice exposed to
 a relevant MISD of UV radiation from a source **emitting** a UV
 power spectrum similar to that of sunlight. (C) Elsevier Science S.A.
 CC BIOPHYSICS; BIOCHEMISTRY & MOLECULAR BIOLOGY
 ST Author Keywords: contact hypersensitivity; immunoprotection;
 immunosuppression; mice; **sunscreen**; UV

STP KeyWords Plus (R): HUMAN-**SKIN**; IMMUNOLOGICAL-UNRESPONSIVENESS;
ALLOACTIVATING CAPACITY; INDUCED INFLAMMATION; INDUCED SUPPRESSION; IMMUNE
SUPPRESSION; AMINOBENZOIC ACID; SUN PROTECTION; UV EXPOSURE; INDUCTION

RE

Referenced Author (RAU)	Year (RPY)	VOL (RVL)	PG (RPG)	Referenced Work (RWK)
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L41 ANSWER 24 OF 32 SCISEARCH COPYRIGHT 2003 THOMSON ISI
 AN 1998:677988 SCISEARCH
 GA The Genuine Article (R) Number: 115TU
 TI Commercial **sunscreen** lotions prevent ultraviolet radiation-induced depletion of epidermal Langerhans cells in Skh-1 and C3H mice
 AU Beasley D G (Reprint); Montgomery M A; Moloney S J; Edmonds S; Roberts L K
 CS SCHERING PLOUGH HEALTHCARE PROD, RES & DEV, 3030 JACKSON AVE, MEMPHIS, TN 38151 (Reprint)
 CYA USA
 SO PHOTODERMATOLOGY PHOTOIMMUNOLOGY & PHOTOMEDICINE, (JUN-AUG 1998) Vol. 14, No. 3-4, pp. 90-99.
 Publisher: MUNKSGAARD INT PUBL LTD, 35 NORRE SOGADE, PO BOX 2148, DK-1016 COPENHAGEN, DENMARK.
 ISSN: 0905-4383.
 DT Article; Journal
 FS CLIN
 LA English
 REC Reference Count: 43
 AB There is much controversy regarding the ability of **sunscreens** to prevent ultraviolet (UV)-induced immune suppression. Epidermal Langerhans cells (LC) play a key antigen-presenting role in the afferent limb of the immune system's response to antigens introduced through the **skin**. It has been suggested that depletion of LC in W-exposed **skin** is a critical step toward the induction of immunosuppression by UV radiation. There are a number of disparate reports with inconsistent results concerning the ability of **sunscreens** to prevent UV-induced depletion of LC. The purpose of this study was to systematically evaluate the ability of **sunscreens** to prevent UV-induced LC depletion in mice. Epidermal sheets obtained from **skin** biopsies taken from mice exposed to UV radiation from Kodacel-filtered FS20 sunlamps, which do not **emit** UV power at wavelengths <290 MI, were immunoperoxidase stained for LC using a rat monoclonal antibody against mouse Ia (major histocompatibility complex class II antigen). Time course and dose-response curves for LC depletion were generated for Skh-1 and C3H mice. Dose-response curves for acute UV exposure induced depletion of LC in Skh-1 and C3H mice were similar, but not identical. LC density in the **skin** of Skh-1 mice that received chronic UV exposure (3 days/week for 8 weeks) was reduced by 62% after 2 weeks of exposure, but returned to normal levels by 6 weeks. Five commercial **sunscreen** lotions with labeled sun protection factors (SPF) of 4, 8, 15, 30 and 45 were tested for their capacity to block W-induced depletion of LC. LC were depleted similar to 75% in the **skin** of unprotected or placebo lotion treated Skh-1 mice exposed to UV given on two consecutive days. Conversely, LC depletion was prevented in similarly UV exposed Skh-1 mice protected with a SPF 30 **sunscreen**. In C3H mice the levels of protection against LC depletion provided by the five **sunscreens** were proportional to the level of protection predicted by their labeled SPE Comparisons of dose-response curves showed that significantly higher doses of UV were required for LC depletion and induction of **skin** edema than for the induction of local suppression of contact hypersensitivity. Thus, at UV doses where **sunscreens** provide complete protection against immunosuppression of contact hypersensitivity, prevention of LC depletion and **skin** edema would be expected.
 CC DERMATOLOGY & VENEREAL DISEASES
 ST Author Keywords: ultraviolet rays; **skin**; immunosuppression; Langerhans cells; **sunscreen**
 STP KeyWords Plus (R): CONTACT HYPERSENSITIVITY; HUMAN-**SKIN**; INDUCED SUPPRESSION; INDUCED IMMUNOSUPPRESSION; ALLOACTIVATING CAPACITY; INDUCED INFLAMMATION; IMMUNE SUPPRESSION; AMINOBENZOIC ACID; SUN PROTECTION; LIGHT
 RE Referenced Author | Year | VOL | PG | Referenced Work

(RAU)

| (RPY) | (RVL) | (RPG) |

(RWK)

L41 ANSWER 23 OF 32 SCISEARCH COPYRIGHT 2003 THOMSON ISI
 AN 2002:800457 SCISEARCH
 GA The Genuine Article (R) Number: 594KK
 TI Tanning salons in southwest Poland: a survey of safety standards and professional knowledge of the staff
 AU Szepietowski J C (Reprint); Nowicka D; Soter K; Strzelecka E; Kozera M; Salomon J
 CS Univ Med Wroclaw, Dept Dermatol & Venereol, Ul Chalubinskiego 1, PL-50368 Wroclaw, Poland (Reprint); Univ Med Wroclaw, Dept Dermatol & Venereol, PL-50368 Wroclaw, Poland
 CYA Poland
 SO PHOTODERMATOLOGY PHOTOIMMUNOLOGY & PHOTOMEDICINE, (AUG 2002) Vol. 18, No. 4, pp. 179-182.
 Publisher: BLACKWELL MUNKSGAARD, 35 NORRE SOGADE, PO BOX 2148, DK-1016 COPENHAGEN, DENMARK.
 ISSN: 0905-4383.
 DT Article; Journal
 LA English
 REC Reference Count: 6
 AB Background. Nowadays, sunbeds are very popular worldwide; however - in the majority of countries, including Poland - there are no general guidelines and/or regulations for sunbed usage.
 Purpose: The aim of the study was to evaluate tanning salons in southwest Poland, paying special attention to safety regulations and basic professional knowledge of the staff members of these units. The hypothesis was put that tanning salons offer better **skin** and eye protection if their employees are more educated.
 Methods: Fifty-five sunbed units were visited by the investigators, acting as potential clients. Information was obtained from the staff members in response to previously designed questionnaire.
 Results: Employees of 41.8% of sunbed units had adequate knowledge of radiation used in their establishments. Goggles were provided in 72.7%, sunglasses in additional 25.5% of salons. Lotions with **sunscreens** were available in 41.8% of units. Brief medical history of the client was taken in 20% of units. No dermatological examination was performed. Tanning salons, where employed staff were well-orientated in ultraviolet radiation **emitted** in their units, significantly more frequently recommended usage of goggles and **sunscreens** ($P = 0.0037$ and $P = 0.0033$, respectively). Moreover, in these establishments **sunscreens** were more commonly available ($P = 0.0029$).
 Conclusions: The knowledge of staff members of tanning salons is poor and the eye and **skin** protection is not enough. The results point out the importance of the knowledge of the staff members in providing **skin** and eye protection.
 CC DERMATOLOGY & VENEREAL DISEASES
 ST Author Keywords: eye protection; **skin** protection; sunbeds; tanning; UVA; UVB
 STP KeyWords Plus (R): AREA SURVEY; RISKS
 RE
 Referenced Author | Year | VOL | PG | R

L41 ANSWER 22 OF 32 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
 AN 82118071 EMBASE
 DN 1982118071
 TI Solar urticaria. Determinations of action and inhibition spectra.
 AU Hasei K.; Ichihashi M.
 CS Dept. Dermatol., Kobe Univ. Sch. Med., 650 Kobe, Japan
 SO Archives of Dermatology, (1982) 118/5 (346-350).
 CODEN: ARDEAC
 CY United States
 DT Journal
 FS 037 Drug Literature Index
 013 Dermatology and Venereology
 026 Immunology, Serology and Transplantation
 LA English
 AB A 42-year-old woman acquired solar urticaria approximately ten minutes after exposure to sunlight. Urticaria developed from visible light emitted from a projector lamp after a similar time lag. Monochromatic rays between 400 and 500 nm induced immediate urticaria by irradiation, with four times the minimal urticarial dose. Urticaria that was induced by monochromatic rays or the projector lamp was completely inhibited by immediate reirradiation of test sites with light waves longer than 530 nm. Radiant heat exposure from an electric hair dryer at 50.degree. C had no suppressive effects on the development of urticarial lesions.
 CT Medical Descriptors:
 *homochlorcyclizine
 *photoallergy
 *solar urticaria
 *ultraviolet radiation
 *urticaria
 clinical study
 induction
 inhibition
 diagnosis
 case report
 therapy
 topical drug administration
 drug comparison
 Drug Descriptors:
 *beta carotene
 *bithionol
 *hexachlorophene
 *hydroxyzine
 *indometacin
 *reserpine
 *skin protective agent
 *sunscreen
 *tetrachlorsalan
 *tranexamic acid
 *tribromsalan
 aminosalicyclic acid
 isoniazid

L41 ANSWER 21 OF 32 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.

AN 89055481 EMBASE

DN 1989055481

TI The lives of pigment cells.

AU Nordlund J.J.

CS Department of Dermatology, University of Cincinnati College of Medicine,
Cincinnati, OH 45267, United States

SO Clinics in Geriatric Medicine, (1989) 5/1 (91-108).

ISSN: 0749-0690 CODEN: CGMEE6

CY United States

DT Journal

FS 002 Physiology

013 Dermatology and Venereology

020 Gerontology and Geriatrics

LA English

SL English

AB It is common knowledge that melanocytes reside in the **skin** and in the eyes. A popular misconception is that the only function of pigment cells is to provide for the **skin** and eyes a shield against visible and ultraviolet radiation **emitted** by the sun. Indeed, the cells synthesize melanin, which does absorb most electromagnetic irradiation, albeit not efficiently. However, melanocytes and melanin have other important functions both during embryogenesis and during extrauterine life. Like all cells in the body, melanocytes are subject to the biological enigma called aging. The defects associated with aging (the loss of number and function of cells) seem to affect the pigmentary system. This loss of melanocytes and the decreased function are observable in the graying of hair, the loss of nevi with age, and the smaller quantity of melanin within the eyes and other tissues. We will trace the lives of pigment cells beginning at their origin in the neural crest during the first weeks of embryogenesis, through fetal life, and through the extrauterine existence of the human being. The evolutionary value of the pigmentary system has been the subject of consideration for many thoughtful biologist and recently has been reviewed. It is likely that melanin functions in many ways, for example, as a **sunscreen**, as an oxygen scavenger, as a by-product for detoxification of tyrosine and cysteine radicals, as an embryologic inducer, and possibly as a toxin responsible for the induction of **skin** cancers such as melanomas. Some of these functions will be re-emphasized later.

CT Medical Descriptors:

*choroid

*eye

*hair

*melanocyte

*neural crest

*pigment cell

***skin**

age

aged

short survey

human

L41 ANSWER 20 OF 32 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
 AN 90088741 EMBASE
 DN 1990088741
 TI The effect of short-term application of PABA on photocarcinogenesis.
 AU Flindt-Hansen H.; Thune P.; Eeg-Larsen T.
 CS Department of Dermatology, Ullevaal Hospital, N-0407 Oslo 4, Norway
 SO Acta Dermato-Venereologica, (1990) 70/1 (72-75).
 ISSN: 0001-5555 CODEN: ADVEA4
 CY Sweden
 DT Journal; Article
 FS 013 Dermatology and Venereology
 016 Cancer
 LA English
 SL English
 AB Photocarcinogenesis was induced in 90 lightly-pigmented hairless mice using a Philips Tl 40 W/12 light source which **emits** mainly UVB (290-320 nm). During one-third of the induction period (weeks 16-26) a group of 30 mice were protected by topical para-aminobenzoic acid (PABA) and then irradiated again without protection up to week 30 and observed for a further 10 weeks. The application of PABA resulted in a significant delay ($p < 0.05$) in tumour induction and discontinuation of PABA caused an abrupt decline in the number of tumour-free animals. At the end of the study there was a significant difference in the yield of carcinomas for the PABA group, 20, compared with 78 for non-protected mice ($p < 0.05$). There was also a statistically significant difference ($p < 0.05$) between the weight of dorsal **skin** in non-protected mice compared with the PABA-protected group, the latter showing no difference from a control group of non-irradiated mice. The proportion of benign tumours in the PABA group was significantly ($p < 0.05$) higher than in the non-protected group, suggesting an inhibition of the photocarcinogenic process.
 CT Medical Descriptors:
 *radiation carcinogenesis
 ***skin cancer**
 *ultraviolet radiation
 mouse
 animal experiment
 animal cell
 nonhuman
 article
 priority journal
 Drug Descriptors:
 ***sunscreen**
 4 aminobenzoic acid
 RN (4 aminobenzoic acid) 150-13-0

L41 ANSWER 19 OF 32 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
 AN 90140539 EMBASE
 DN 1990140539
 TI Photocarcinogenesis is retarded by a partly photodegraded solution of
 para-aminobenzoic acid.
 AU Flindt-Hansen H.; Thune P.; Nielsen C.J.
 CS Department of Dermatology, Ullevaal Hospital, N-0407 Oslo 4, Norway
 SO Photodermatology, (1989) 6/6 (263-267).
 ISSN: 0108-9684 CODEN: PHTDEI
 CY Denmark
 DT Journal; Article
 FS 013 Dermatology and Venereology
 016 Cancer
 030 Pharmacology
 037 Drug Literature Index
 LA English
 SL English
 AB A solution of para-aminobenzoic acid (PABA) was exposed to ultraviolet
 (UV) radiation **emitted** from a Philips TL 40 W/12 sunlamp and the
 degree of photodegradation following an exposure of 27 J/cm2 was estimated
 to be approximately 40%. The formation of the photoproducts was confirmed
 by mass spectroscopy and UV spectroscopy. The solution was painted on the
 backs of hairless light-pigmented mice prior to daily UV irradiation by
 the above sunlamp, and this procedure was continued for 30 weeks. The
 preirradiated solution of PABA significantly retarded the tumor induction
 time and reduced significantly the number of squamous cell carcinomas
 compared with non-protected controls. This tumor-retarding ability did not
 differ significantly from the effect achieved then using nonirradiated
 PABA.
 CT Medical Descriptors:
 *photodegradation
 *radiation carcinogenesis
 ***skin cancer**
 *ultraviolet radiation
 mouse
 animal experiment
 animal cell
 nonhuman
 article
 Drug Descriptors:
 ***sunscreen**
 *4 aminobenzoic acid
 RN (4 aminobenzoic acid) 150-13-0

L41 ANSWER 18 OF 32 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
 AN 90251342 EMBASE
 DN 1990251342
 TI [Study on the effectiveness of a **sunscreen** containing ethoxy
 cynamate and dibenzoylmethane].
 STUDIO SULL'EFFICACIA DI UNO SCHERMO SOLARE A BASE DI METOSSICINNAMATO E
 DIBENZOILMETANO.
 AU Procaccini E.M.; Criscuolo I.; Perrelli P.; Monfrecola G.
 CS Clinica Dermatologica, II Facolta di Medicina, Via S. Pansini 5, 80131
 Napoli, Italy
 SO Annali Italiani di Dermatologia Clinica e Sperimentale, (1990) 44/1
 (25-29).
 ISSN: 0003-4703 CODEN: ADCRAG
 CY Italy
 DT Journal; Article
 FS 013 Dermatology and Venereology
 037 Drug Literature Index
 LA Italian
 SL English
 AB The effectiveness of a **sunscreen** containing methoxy cynamate
 and dibenzoylmethane has been evaluated in 12 healthy volunteers. For each
 subject, two symmetric areas of bottoms (one of them previously receiving
 the **sunscreen**) have been irradiated with a bank of three 400 W
 metal halide lamps, **emitting** in the UVB range (max at 300 nm),
 in order to detect the Minimal Erythema Dose (MED). The MED values have
 been determined by a color analyzer. The results have shown that, at least
 in the UVB range, the MED values of the **sunscreen**-protected
skin areas were 3-8 fold higher than that of the unprotected
 areas.
 CT Medical Descriptors:
 human
 normal human
 human experiment
 article
 Drug Descriptors:
 ***sunscreen**
 *4 methoxycinnamic acid 2 ethylhexyl ester
 *avobenzene
 RN (4 methoxycinnamic acid 2 ethylhexyl ester) 5466-77-3; (avobenzene)
 70356-09-1

L41 ANSWER 17 OF 32 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
 AN 97206026 EMBASE
 DN 1997206026
 TI **Sunscreens** prevent local and systemic immunosuppression of contact hypersensitivity in mice exposed to solar-simulated ultraviolet radiation.
 AU Roberts L.K.; Beasley D.G.
 CS L.K. Roberts, Advanced Product Research, AR-3-59, Schering-Plough HealthCare Products, 3030 Jackson Avenue, Memphis, TN 38151, United States
 SO Journal of Photochemistry and Photobiology B: Biology, (1997) 39/2 (121-129).
 Refs: 46
 ISSN: 1011-1344 CODEN: JPPBEG
 PUI S 1011-1344(97)00003-1
 CY Switzerland
 DT Journal; Article
 FS 013 Dermatology and Venereology
 026 Immunology, Serology and Transplantation
 030 Pharmacology
 037 Drug Literature Index
 039 Pharmacy
 LA English
 SL English
 AB Ultraviolet (UV) irradiation causes the immunosuppression of contact hypersensitivity (CH) responses in animals and humans. There are conflicting reports regarding the effectiveness of **sunscreens** in preventing UV-induced suppression of both local-type CH (induced by the application of the contact sensitizer directly to UV-exposed **skin**) and systemic-type CH (induced by the application of the contact sensitizer to an unirradiated **skin** site 3 days after UV exposure). The purposes of this study were as follows: 1. to derive solar simulator UV dose-response curves for the induction of local and systemic CH suppression in C3H mice; 2. to establish minimum immune suppression doses (MISDs) for local and systemic CH; 3. to determine the local and systemic immune protection capacity of two commercial **sunscreen** lotions with labeled sun protection factors (SPFs) of 4 and 8. Dose-response curves for the induction of local and systemic CH suppression were derived by exposing groups of mice to a range of full-spectrum UV doses (0.37-21.4 kJ m⁻²) on two consecutive days delivered from a filtered 1000 W xenon arc lamp solar simulator. The MISDs, defined as the lowest dose tested to cause approximately 50% suppression of the normal CH response, were obtained from the dose-response curves. Although the local and systemic immunosuppression dose-response curves were not statistically different, the MISD for local suppression of CH (1.35 kJ m⁻²) was about fivefold lower than that for systemic CH suppression (6.76 kJ m⁻²). The MISD was used as the endpoint to determine **sunscreen** immune protection levels. Both **sunscreens**, applied at 2 mg cm⁻², provided immune protection against the induction of local and systemic CH suppression in mice exposed to an effective UV dose of 1 MISD given through the **sunscreen**, i.e. 4 MISD to SPF 4 **sunscreen**-protected mice and 8 MISD to SPF 8 **sunscreen**-protected mice mounted CH responses that were significantly greater than those elicited in unprotected mice exposed to 1 MISD of solar-simulated UV radiation. The calculated immune protection factors for these **sunscreens** exceeded the level of protection predicted by their labeled SPFs, i.e. the local immune protection factor of both **sunscreens** was 15 and the systemic immune protection factors were 8 for the SPF 4 **sunscreen** and 15 for the SPF 8 **sunscreen**. Our data show that these two **sunscreens** provide levels of immune protection which exceed the levels predicted by their labeled SPFs in immunoprotection tests conducted in mice exposed to a relevant MISD of UV radiation from a source **emitting** a UV power spectrum similar to that of sunlight.

CT Medical Descriptors:
*contact allergy
*immune deficiency
*solar radiation
animal experiment
article
controlled study
dose response
drug formulation
female
mouse
nonhuman
priority journal
topical drug administration
ultraviolet radiation
Drug Descriptors:
 *sunscreen: DV, drug development
 *sunscreen: PR, pharmaceuticals
4 methoxycinnamic acid 2 ethylhexyl ester: DV, drug development
4 methoxycinnamic acid 2 ethylhexyl ester: PR, pharmaceuticals
coppertone: DV, drug development
coppertone: PR, pharmaceuticals
oxybenzone: DV, drug development
oxybenzone: PR, pharmaceuticals
RN (4 methoxycinnamic acid 2 ethylhexyl ester) 5466-77-3; (oxybenzone)
131-57-7
CN (1) Coppertone
CO (1) Schering plough (United States)

L41 ANSWER 16 OF 32 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
 AN 1998263802 EMBASE
 TI [Solar simulators and sunlight].
 SIMULATEURS SOLAIRES ET SOLEIL STANDARD.
 AU Chardon A.
 CS A. Chardon, L'Oreal, Recherche Appliquee et Developpement, 92110 Clichy,
 France
 SO Nouvelles Dermatologiques, (1998) 17/5 (330-335).
 Refs: 22
 ISSN: 0752-5370 CODEN: NODEE2
 CY France
 DT Journal; Article
 FS 013 Dermatology and Venereology
 027 Biophysics, Bioengineering and Medical Instrumentation
 LA French
 SL English; French
 AB The solar simulator, based on a filtered xenon lamp to simulate the total
 ultraviolet spectrum of the sunlight, were originally developed in the USA
 about thirty years ago. They are widely used for the determination of the
 Sun Protection Factor (SPF) of **sunscreens** or for photobiological
 studies. Now, it has been suggested that the spectrum **emitted** by
 such simulators was not accurately balanced to the detriment of its
 content in UVA energy, which may induce a significant bias in the
 evaluation of the **sunscreens** efficacy: SPF, photostability, UVA
 protection. The aim of this study was to compare the characteristics of
 solar simulator spectra with realistic solar irradiance conditions, in
 order to determine the standard values to be respected and the
 corresponding UV doses to be applied per MED unit in photostability
 testing. The key results indicate that in quasi-zenithal conditions of sun
 exposure (limit conditions) at sea level air mass 1) the mean UV, UVB and
 UVA fluxes are respectively about 5.9, 0.31 and 5.6 mW/cm² with a UVA/UVB
 ratio of 18.3/1, while the ratio of the erythema effective fluxes
 UVBe/UVAe is 4.7/1; thus, about 83% of the erythema is due to the UVB rays
 and 17% to the UVA. In more temperature conditions, for a sun height above
 the horizon of 42.degree. (air mass 1.5), the UVA/UVB ratio is then higher
 than 20/1 and about 25% of the erythema is due to the UVA rays. The
 quantity of total UV energy delivered with one standard MED (21 mJ/cm²e)
 is 5.6 J/cm² in the zenithal sun, and about 8 J/cm² in a the temperature
 sun. With the usual solar simulators (xenon filtered with WG320/1 mm) the
 ratio of the erythema effective fluxes UVBe/UVAe is higher than 10/1; in
 these conditions, less than 10% of the erythema is due to the UVA and the
 total UV dose applied per MED is only about 2 J/cm², i.e. three times less
 than that received in the actual sun. Thus, the output spectrum of usual
 solar simulators is not representative of realistic conditions of sun
 exposure, because of an excess of UVB erythema efficacy and/or a defect
 of UVA energy. As indicative data, a minimal thickness of 1.5 mm WG320
 filter is generally necessary as short cut-off filter to better mimic the
 sun. But, the recommendation of such a filtering system is not sufficient:
 every solar simulator should be checked by spectroradiometry in definite
 conditions and the optical system adapted for the characteristics of the
 output spectrum to closely approach the nominal values of the 'standard
 sun' according to the Colipa SPF test method, without exceeding its UVB
 relative content.
 CT Medical Descriptors:
 *solar radiation
 *skin protection
 simulator
 ultraviolet b radiation
 ultraviolet a radiation
 radiation dose
 erythema
 sunlight
 spectral sensitivity

article

L41 ANSWER 13 OF 32 MEDLINE
 AN 85165732 MEDLINE
 DN 85165732 PubMed ID: 6531284
 TI UVA sensitivity and topical photoprotection in polymorphous light eruption.
 AU McFadden N
 SO PHOTO-DERMATOLOGY, (1984 Apr) 1 (2) 76-8.
 Journal code: 8407997. ISSN: 0108-9684.
 CY Denmark
 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals
 EM 198504
 ED Entered STN: 19900320
 Last Updated on STN: 19900320
 Entered Medline: 19850429
 AB The efficacy of a non-PABA, non-benzophenone broad-spectrum **sunscreen** was investigated experimentally in 8 patients with polymorphous light eruption (PMLE). A UVA-SUN 2000 lamp, **emitting** high intensity UVA radiation was used to irradiate unprotected and **sunscreen** protected **skin** sites of the upper back of each patient. Morphological and histological **skin** changes were noted in non-protected test-sites, while no clinical or microscopic changes were observed in the **sunscreen**-treated test-sites. Low minimal erythema dose (MED) values for both UVA and UVB light were noted in several PMLE patients.
 CT Check Tags: Female; Human; Male
 *Photosensitivity Disorders: PC, prevention & control
 *Sunscreening Agents: TU, therapeutic use
 *Ultraviolet Rays: AE, adverse effects
 CN 0 (Sunscreening Agents)

 L41 ANSWER 14 OF 32 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
 AN 2002334206 EMBASE
 TI Tanning salons in southwest Poland: A survey of safety standards and professional knowledge of the staff.
 AU Szepletowski J.C.; Nowicka D.; Soter K.; Strzelecka E.; Kozera M.; Salomon J.
 CS Dr. J.C. Szepletowski, Department of Dermatol./Venereology, University of Medicine, Ul. Chalubinskiego 1, 50-368 Wroclaw, Poland.
 jszeplet@derm.am.wroc.pl
 SO Photodermatology Photoimmunology and Photomedicine, (2002) 18/4 (179-182).
 Refs: 6
 ISSN: 0905-4383 CODEN: PPPHEW
 CY Denmark
 DT Journal; Article
 FS 013 Dermatology and Venereology
 037 Drug Literature Index
 LA English
 SL English
 AB Background: Nowadays, sunbeds are very popular worldwide; however - in the majority of countries, including Poland - there are no general guidelines and/or regulations for sunbed usage. Purpose: The aim of the study was to evaluate tanning salons in southwest Poland, paying special attention to safety regulations and basic professional knowledge of the staff members of these units. The hypothesis was put that tanning salons offer better **skin** and eye protection if their employees are more educated. Methods: Fifty-five sunbed units were visited by the investigators, acting as potential clients. Information was obtained from the staff members in response to previously designed questionnaire. Results: Employees of 41.8% of sunbed units had adequate knowledge of radiation used in their establishments. Goggles were provided in 72.7%, sunglasses in additional 25.5% of salons. Lotions with **sunscreens** were available in 41.8% of units. Brief medical history of the client was taken in 20% of units.

No dermatological examination was performed. Tanning salons, where employed staff were well-orientated in ultraviolet radiation **emitted** in their units, significantly more frequently recommended usage of goggles and **sunscreens** (P = 0.0037 and P = 0.0033, respectively). Moreover, in these establishments **sunscreens** were more commonly available (P = 0.0029). Conclusions: The knowledge of staff members of tanning salons is poor and the eye and **skin** protection is not enough. The results point out the importance of the knowledge of the staff members in providing **skin** and eye protection.

CT Medical Descriptors:
*ultraviolet radiation
*radiation protection
*eye protection
 ***skin protection**
Poland
health survey
safety
standard
staff training
hypothesis
employee
health education
medical information
questionnaire
eye protective device
lotion
anamnesis
dermatological procedures
human
controlled study
article
priority journal
Drug Descriptors:
 sunscreen

L41 ANSWER 15 OF 32 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
AN 1998301920 EMBASE
TI Commercial **sunscreen** lotions prevent ultraviolet radiation-induced depletion of epidermal Langerhans cells in Skh-1 and C3H mice.
AU Beasley D.G.; Montgomery M.A.; Moloney S.J.; Edmonds S.; Roberts L.K.
CS D.G. Beasley, Research and Development, Schering-Plough HealthCare Products, 3030 Jackson Avenue, Memphis, TN 38151, United States
SO Photodermatology Photoimmunology and Photomedicine, (1998) 14/3-4 (90-99).
Refs: 43
ISSN: 0905-4383 CODEN: PPPHEW
CY Denmark
DT Journal; Article
FS 013 Dermatology and Venereology
 046 Environmental Health and Pollution Control
LA English
SL English
AB There is much controversy regarding the ability of **sunscreens** to prevent ultraviolet (UV)-induced immune suppression. Epidermal Langerhans cells (LC) play a key antigen-presenting role in the afferent limb of the immune system's response to antigens introduced through the **skin**. It has been suggested that depletion of LC in UV-exposed **skin** is a critical step toward the induction of immunosuppression by UV radiation. There are a number of disparate reports with inconsistent results concerning the ability of **sunscreens** to prevent UV-induced depletion of LC. The purpose of this study was to systematically evaluate the ability of **sunscreens** to prevent UV-induced LC depletion in mice. Epidermal sheets obtained from **skin**

biopsies taken from mice exposed to UV radiation from Kodacel-filtered FS20 sunlamps, which do not **emit** UV power at wavelengths <290 nm, were immunoperoxidase stained for LC using a rat monoclonal antibody against mouse Ia (major histocompatibility complex class II antigen). Time course and dose-response curves for LC depletion were generated for Skh-1 and C3H mice. Dose-response curves for acute UV exposure induced depletion of LC in Skh-1 and C3H mice were similar, but not identical. LC density in the **skin** of Skh-1 mice that received chronic UV exposure (3 days/week for 8 weeks) was reduced by 62% after 2 weeks of exposure, but returned to normal levels by 6 weeks. Five commercial **sunscreen** lotions with labeled sun protection factors (SPF) of 4, 8, 15, 30 and 45 were tested for their capacity to block UV-induced depletion of LC. LC were depleted .apprx.75% in the **skin** of unprotected or placebo lotion treated Skh-1 mice exposed to UV given on two consecutive days. Conversely, LC depletion was prevented in similarly UV exposed Skh-1 mice protected with a SPF 30 **sunscreen**. In C3H mice the levels of protection against LC depletion provided by the five **sunscreens** were proportional to the level of protection predicted by their labeled SPF. Comparisons of dose-response curves showed that significantly higher doses of UV were required for LC depletion and induction of **skin** edema than for the induction of local suppression of contact hypersensitivity. Thus, at UV doses where **sunscreens** provide complete protection against immunosuppression of contact hypersensitivity, prevention of LC depletion and **skin** edema would be expected.

CT Medical Descriptors:

- *langerhans cell
- *immune deficiency
- *skin edema**
- *contact allergy
- lotion
- ultraviolet radiation
- radiation protection
- skin protection**
- skin biopsy**
- radiation dose
- nonhuman
- female
- mouse
- animal experiment
- animal model
- controlled study
- animal tissue
- article
- priority journal

Drug Descriptors:

- *sunscreen**
- Ia antigen: EC, endogenous compound
- major histocompatibility antigen class 2: EC,

L41 ANSWER 12 OF 32 MEDLINE
 AN 96242939 MEDLINE
 DN 96242939 PubMed ID: 8992508
 TI Ultraviolet spectral energy differences affect the ability of
sunscreen lotions to prevent ultraviolet-radiation-induced
 immunosuppression.
 AU Roberts L K; Beasley D G; Learn D B; Giddens L D; Beard J; Stanfield J W
 CS Schering-Plough HealthCare Products, Memphis, TN 38151, USA.
 SO PHOTOCHEMISTRY AND PHOTOBIOLOGY, (1996 Jun) 63 (6) 874-84.
 Journal code: 0376425. ISSN: 0031-8655.
 CY United States
 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals
 EM 199701
 ED Entered STN: 19970128
 Last Updated on STN: 19970128
 Entered Medline: 19970116
 AB Acute exposure to UV radiation causes immunosuppression of contact
 hypersensitivity (CH) responses. Past studies conducted with unfiltered
 sunlamps **emitting** nonsolar spectrum UV power (wavelengths below
 295 nm) or using excessive UV doses have suggested **sunscreens**
 may not prevent UV-induced immunosuppression in mice. This study was thus
 designed to evaluate critically the effects of different UV energy spectra
 on the immune protection capacity of **sunscreen** lotions. Minimum
 immune suppression doses (MISD), i.e. the lowest UV dose to cause
 approximately 50% suppression of the CH response to dinitrofluorobenzene
 in C3H mice, were established for three artificial UV sources. The MISD
 for each UV source was 0.25 kJ/m² for unfiltered FS20 sunlamps (FS), 0.90
 kJ/m² for Kodacel-filtered FS20 sunlamps (KFS), which do not **emit**
 UV power at wavelengths < 290 nm, and 1.35 kJ/m² for a 1000 W filtered
 xenon arc lamp solar simulator. Using MISD as baseline,
sunscreens with labeled sun protection factors (SPF) of 4, 8, 15
 and 30 were tested with each UV source to establish their relative immune
 protection factors. The immune protection factor of each
sunscreen exceeded its labeled SPF in tests conducted with the
 solar simulator, which has a UV power spectrum (295-400 nm) similar to
 that of sunlight. Conversely, **sunscreen** immune protection
 factors were significantly less than the labeled SPF in tests conducted
 with FS and KFS. Comparison of the immunosuppression effectiveness
 spectra showed that relatively small amounts of nonsolar spectrum UV
 energy, i.e. UVC (200-290 nm) and/or shorter wavelength UVB (between 290
 and 295 nm), produced by FS and KFS contributes significantly to the
 induction of immunosuppression. For example, 36.3% and 3.5% of the total
 immunosuppressive UV energy from FS and KFS, respectively, lies below 295
 nm. **Sunscreen** absorption spectra showed that transmission of
 immunosuppressive UV energy below 295 nm for FS was at least eight-fold
 higher than that for KFS. Compared to the solar simulator UV spectrum the
 transmission of nonsolar immunosuppressive UV energy through
sunscreens was > 15-fold higher for FS and > or = 1.5-fold higher
 for KFS. These data demonstrate that relevant evaluations of
sunscreen immune protection can only be obtained when tests are
 conducted with UV sources that produce UV power spectra similar to that of
 sunlight and UV doses are employed that are based on established MISD.
 CT Check Tags: Animal; Female
 Dermatitis, Contact: PC, prevention & control
 Dose-Response Relationship, Radiation
 *Immune Tolerance: DE, drug effects
 *Immune Tolerance: RE, radiation effects
 Mice
 Mice, Inbred C3H
 Photobiology
 Skin: DE, drug effects

Skin: IM, immunology

Skin: RE, radiation effects

*Sunscreening Agents: PD, pharmacology

*Ultraviolet Rays: AE, adverse effects

CN 0 (Sunscreening Agents)

L41 ANSWER 11 OF 32 MEDLINE
 AN 97368844 MEDLINE
 DN 97368844 PubMed ID: 9225458
 TI **Sunscreens** prevent local and systemic immunosuppression of contact hypersensitivity in mice exposed to solar-simulated ultraviolet radiation.
 AU Roberts L K; Beasley D G
 CS Advanced Product Research, Schering-Plough HealthCare Products, Memphis, TN 38151, USA.
 SO JOURNAL OF PHOTOCHEMISTRY AND PHOTOBIOLOGY. B, BIOLOGY, (1997 Jun) 39 (2) 121-9.
 Journal code: 8804966. ISSN: 1011-1344.
 CY Switzerland
 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals
 EM 199709
 ED Entered STN: 19970916
 Last Updated on STN: 19970916
 Entered Medline: 19970903
 AB Ultraviolet (UV) irradiation causes the immunosuppression of contact hypersensitivity (CH) responses in animals and humans. There are conflicting reports regarding the effectiveness of **sunscreens** in preventing UV-induced suppression of both local-type CH (induced by the application of the contact sensitizer directly to UV-exposed **skin**) and systemic-type CH (induced by the application of the contact sensitizer to an unirradiated **skin** site 3 days after UV exposure). The purposes of this study were as follows: 1. to derive solar simulator UV dose-response curves for the induction of local and systemic CH suppression in C3H mice; 2. to establish minimum immune suppression doses (MISDs) for local and systemic CH; 3. to determine the local and systemic immune protection capacity of two commercial **sunscreen** lotions with labeled sun protection factors (SPFs) of 4 and 8. Dose-response curves for the induction of local and systemic CH suppression were derived by exposing groups of mice to a range of full-spectrum UV doses (0.37-21.4 kJ m⁻²) on two consecutive days delivered from a filtered 1000 W xenon arc lamp solar simulator. The MISDs, defined as the lowest dose tested to cause approximately 50% suppression of the normal CH response, were obtained from the dose-response curves. Although the local and systemic immunosuppression dose-response curves were not statistically different, the MISD for local suppression of CH (1.35 kJ m⁻²) was about fivefold lower than that for systemic CH suppression (6.76 kJ m⁻²). The MISD was used as the endpoint to determine **sunscreen** immune protection levels. Both **sunscreens**, applied at 2 mg cm⁻², provided immune protection against the induction of local and systemic CH suppression in mice exposed to an effective UV dose of 1 MISD given through the **sunscreen**, i.e. 4 MISD to SPF 4 **sunscreen**-protected mice and 8 MISD to SPF 8 **sunscreen**-protected mice mounted CH responses that were significantly greater than those elicited in unprotected mice exposed to 1 MISD of solar-simulated UV radiation. The calculated immune protection factors for these **sunscreens** exceeded the level of protection predicted by their labeled SPFs, i.e. the local immune protection factor of both **sunscreens** was 15 and the systemic immune protection factors were 8 for the SPF 4 **sunscreen** and 15 for the SPF 8 **sunscreen**. Our data show that these two **sunscreens** provide levels of immune protection which exceed the levels predicted by their labeled SPFs in immunoprotection tests conducted in mice exposed to a relevant MISD of UV radiation from a source **emitting** a UV power spectrum similar to that of sunlight.
 CT Check Tags: Animal; Female
 *Dermatitis, Contact: IM, immunology
 *Immunosuppression
 Mice

Mice, Inbred C3H

Skin: DE, drug effects

Skin: RE, radiation effects

*Sunscreening Agents: PD, pharmacology

*Ultraviolet Rays

CN 0 (Sunscreening Agents)

L41 ANSWER 9 OF 32 MEDLINE
 AN 2002633042 MEDLINE
 DN 22279039 PubMed ID: 12390672
 TI Tanning salons in southwest Poland: a survey of safety standards and professional knowledge of the staff.
 AU Szepietowski Jacek C; Nowicka Danuta; Soter Katarzyna; Strzelecka Ewa; Kozera Marzena; Salomon Joanna
 CS Department of Dermatology and Venereology, University of Medicine, Wroclaw, Poland.. jszepiet@derm.am.wroc.pl
 SO PHOTODERMATOLOGY, PHOTOIMMUNOLOGY AND PHOTOMEDICINE, (2002 Aug) 18 (4) 179-82.
 Journal code: 9013641. ISSN: 0905-4383.
 CY Denmark
 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals
 EM 200212
 ED Entered STN: 20021023
 Last Updated on STN: 20021220
 Entered Medline: 20021219
 AB BACKGROUND: Nowadays, sunbeds are very popular worldwide; however--in the majority of countries, including Poland--there are no general guidelines and/or regulations for sunbed usage. PURPOSE: The aim of the study was to evaluate tanning salons in southwest Poland, paying special attention to safety regulations and basic professional knowledge of the staff members of these units. The hypothesis was put that tanning salons offer better **skin** and eye protection if their employees are more educated. METHODS: Fifty-five sunbed units were visited by the investigators, acting as potential clients. Information was obtained from the staff members in response to previously designed questionnaire. RESULTS: Employees of 41.8% of sunbed units had adequate knowledge of radiation used in their establishments. Goggles were provided in 72.7%, sunglasses in additional 25.5% of salons. Lotions with **sunscreens** were available in 41.8% of units. Brief medical history of the client was taken in 20% of units. No dermatological examination was performed. Tanning salons, where employed staff were well-orientated in ultraviolet radiation **emitted** in their units, significantly more frequently recommended usage of goggles and **sunscreens** ($P = 0.0037$ and $P = 0.0033$, respectively). Moreover, in these establishments **sunscreens** were more commonly available ($P = 0.0029$). CONCLUSIONS: The knowledge of staff members of tanning salons is poor and the eye and **skin** protection is not enough. The results point out the importance of the knowledge of the staff members in providing **skin** and eye protection.
 CT Check Tags: Human
 Adolescent
 Adult
 *Beauty Culture
 Child
 Data Collection
 Educational Status
 Eye Protective Devices
 Poland
 Prospective Studies
 Radiation Protection
 Safety
 *Skin: RE, radiation effects
 Sunlight
 Sunscreening Agents: AD, administration & dosage
 *Ultraviolet Rays: AE, adverse effects
 CN 0 (Sunscreening Agents)
 L41 ANSWER 10 OF 32 MEDLINE

AN 1998452531 MEDLINE
 DN 98452531 PubMed ID: 9779495
 TI Commercial **sunscreen** lotions prevent ultraviolet radiation-induced depletion of epidermal Langerhans cells in Skh-1 and C3H mice.
 AU Beasley D G; Montgomery M A; Moloney S J; Edmonds S; Roberts L K
 CS Schering-Plough HealthCare Products, Memphis, TN 38151, USA.
 SO PHOTODERMATOLOGY, PHOTOIMMUNOLOGY AND PHOTOMEDICINE, (1998 Jun-Aug) 14 (3-4) 90-9.
 Journal code: 9013641. ISSN: 0905-4383.
 CY Denmark
 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals
 EM 199812
 ED Entered STN: 19990115
 Last Updated on STN: 19990115
 Entered Medline: 19981221
 AB There is much controversy regarding the ability of **sunscreens** to prevent ultraviolet (UV)-induced immune suppression. Epidermal Langerhans cells (LC) play a key antigen-presenting role in the afferent limb of the immune system's response to antigens introduced through the **skin**. It has been suggested that depletion of LC in UV-exposed **skin** is a critical step toward the induction of immunosuppression by UV radiation. There are a number of disparate reports with inconsistent results concerning the ability of **sunscreens** to prevent UV-induced depletion of LC. The purpose of this study was to systematically evaluate the ability of **sunscreens** to prevent UV-induced LC depletion in mice. Epidermal sheets obtained from **skin** biopsies taken from mice exposed to UV radiation from Kodacel-filtered FS20 sunlamps, which do not **emit** UV power at wavelengths < 290 nm, were immunoperoxidase stained for LC using a rat monoclonal antibody against mouse Ia (major histocompatibility complex class II antigen). Time course and dose-response curves for LC depletion were generated for Skh-1 and C3H mice. Dose-response curves for acute UV exposure induced depletion of LC in Skh-1 and C3H mice were similar, but not identical. LC density in the **skin** of Skh-1 mice that received chronic UV exposure (3 days/week for 8 weeks) was reduced by 62% after 2 weeks of exposure, but returned to normal levels by 6 weeks. Five commercial **sunscreen** lotions with labeled sun protection factors (SPF) of 4, 8, 15, 30 and 45 were tested for their capacity to block UV-induced depletion of LC. LC were depleted approximately 75% in the **skin** of unprotected or placebo lotion treated Skh-1 mice exposed to UV given on two consecutive days. Conversely, LC depletion was prevented in similarly UV exposed Skh-1 mice protected with a SPF 30 **sunscreen**. In C3H mice the levels of protection against LC depletion provided by the five **sunscreens** were proportional to the level of protection predicted by their labeled SPF. Comparisons of dose-response curves showed that significantly higher doses of UV were required for LC depletion and induction of **skin** edema than for the induction of local suppression of contact hypersensitivity. Thus, at UV doses where **sunscreens** provide complete protection against immunosuppression of contact hypersensitivity, prevention of LC depletion and **skin** edema would be expected.
 CT Check Tags: Animal; Female; In Vitro
 Cell Count
 Dose-Response Relationship, Radiation
 *Epidermis: CY, cytology
 Immunoenzyme Techniques
 Langerhans Cells: CY, cytology
 Langerhans Cells: DE, drug effects
 *Langerhans Cells: RE, radiation effects
 Mice
 Mice, Inbred C3H

Mice, Inbred HRS

Mice, Inbred Strains

*Sunscreening Agents: PD, pharmacology

*Ultraviolet Rays

CN 0 (Sunscreening Agents)

L41 ANSWER 6 OF 32 CAPLUS COPYRIGHT 2003 ACS
 AN 1997:259032 CAPLUS
 DN 126:297453
 TI Broad-spectrum **sunscreens** with UVA I and UVA II absorbers
 provide increased protection against solar-simulating radiation-induced
 dermal damage in hairless mice
 AU Kligman, Lorraine H.; Agin, Patricia P.; Sayre, Robert M.
 CS Department of Dermatology, University of Pennsylvania School of Medicine,
 Philadelphia, PA, 19104-6142, USA
 SO Journal of the Society of Cosmetic Chemists (1996), 47(3), 129-155
 CODEN: JSCCA5; ISSN: 0037-9832
 PB Society of Cosmetic Chemists
 DT Journal
 LA English
 CC 62-4 (Essential Oils and Cosmetics)
 AB Previous expts. designed to examine **sunscreen** protection against
 chronic UV radiation-induced **skin** damage in hairless mice have
 used radiation sources **emitting** mainly UVB or UVA radiation.
 Because humans are exposed to full-spectrum solar radiation, we were
 interested in examg. the efficacy of 3 **sunscreens**, with
 increasing spectral absorption into the UVA range, against chronic
 solar-simulating radiation (SSR). Three groups of hairless mice received
 a cumulative SSR dose of 10 and 16 times a previously detd. minimal
 photoaging dose (MPD) over periods of 18 and 30 wk. Each twice-weekly
 exposure was designed to equal the SPF value of the first
sunscreen, an SPF-7 **sunscreen** contg. the UVB absorber
 octyl methoxycinnamate. The second **sunscreen**, in addn. to the
 UVB absorber, contained a UVA II absorber (oxybenzone) and had an SPF of
 16. The third, with an SPF of 18, contained the UVB and UVA II absorbers
 plus a UVA I absorber (avobenzone). These conditions allowed assessment
 of the effects of UVB and UVA radiation that are normally transmitted
 through all **sunscreens**. Although none of the **sunscreen**
 -treated mice developed erythema, considerable dermal matrix damage
 occurred in the SPF-7 group, with greater damage at 16 MPD than at 10 MPD.
 The SPF-16 **sunscreen** allowed less but clearly recognizable
 damage at both dose points. The SPF-18 **sunscreen** with the
 broadest spectral absorption provided the greatest protection. These
 results support the need for high-SPF broad-spectrum sun-screen protection
 that includes the entire UVA spectrum to reduce photodamage that results
 from chronic exposure to sunlight.
 ST **sunscreen** UVA absorber solar radiation
 IT Erythema
 Skin
 Solar radiation
 Sunscreens
 UV A radiation
 (**sunscreens** with UVA absorbers for protection against solar
 radiation-induced dermal damage)
 IT 131-57-7, Oxybenzone 5466-77-3 70356-09-1, Avobenzone
 RL: BUU (Biological use, unclassified); BIOL (Biological study); USES
 (Uses)
 (**sunscreens** with UVA absorbers for protection against solar
 radiation-induced dermal damage)

L41 ANSWER 7 OF 32 CAPLUS COPYRIGHT 2003 ACS
 AN 1992:37060 CAPLUS
 DN 116:37060
 TI Photocarcinogenesis and consumer product testing: technical aspects
 AU Sambuco, C. P.; Davies, R. E.; Forbes, P. D.; Hoberman, A. M.
 CS Cent. Photobiol., Argus, Inc., Horsham, PA, 19044, USA
 SO Toxicology Methods (1991), 1(1), 75-83
 CODEN: TOMEEB; ISSN: 1051-7235
 DT Journal

LA English
 CC 8-6 (Radiation Biochemistry)
 Section cross-reference(s): 63

AB Photocarcinogenesis is the induction of tumors by sunlight. Since consumer products have been found to modify the course of cutaneous photocarcinogenesis in animal models, and since this modification is thought to be clin. relevant, procedures have been developed and implemented to evaluate a test article/substance as a potential modifier of photocarcinogenesis. Prior to beginning a photocarcinogenesis study, range-finding studies are conducted to detn. certain characteristics of the test article/substance and its vehicle (e.g., primary irritancy, photosensitivity, and photoprotection). Addnl., the doses of test article/substance, the doses of sunlight exposure, and the most appropriate sequence for administering these 2 variables are detd. from the range-finding studies and the intended use of the test article/substance. The test system is the Crl:SKH1 (h/h)BR, albino, hairless mouse. The source of "sunlight" is a 6.5 kW xenon arc lamp modified to **emit** radiation that stimulates sunlight at the earth's surface. Methods for gathering and analyzing tumor data are discussed, and a theor. study is used to facilitate the presentation. Three possible outcomes of this theor. study are presented, and some unique features of the study are given. Any agent that can modify the carcinogenic effect of sunlight should be carefully considered with respect to its benefits and risks.

ST photocarcinogenesis consumer product
 IT **Sunscreens**
 (photocarcinogenesis modification by, testing of)

IT Health physics
 (photocarcinogenesis response to consumer products testing in relation to)

IT Neoplasm
 Skin, neoplasm
 (solar UV radiation induction of, consumer products modification of, testing of)

IT Ultraviolet radiation
 (solar, carcinogenesis induction by, consumer products modification of, testing of)

L41 ANSWER 8 OF 32 CAPLUS COPYRIGHT 2003 ACS
 AN 1985:427050 CAPLUS
 DN 103:27050
 TI A rapid non-injurious method to evaluate the light protective potential of **sunscreens**
 AU Sauermann, Gerhard; Hoppe, Udo
 CS Dev. Cosmed., Beiersdorf A.-G., Hamburg, D-2000, Fed. Rep. Ger.
 SO Journal of the Society of Cosmetic Chemists (1985), 36(2), 125-41
 CODEN: JSCCA5; ISSN: 0037-9832
 DT Journal
 LA English
 CC 62-1 (Essential Oils and Cosmetics)

AB Fluorescing mols. (dansyl residues) within the horny layer **emit** fluorescent light in direct relation to the intensity of the exciting beam. This was used in evaluation of the light protective potential of **sunscreens**. The attenuation of the exciting beam by light-absorbing **sunscreens** covering the surface of the **skin** is followed by a corresponding decrease of **emitted** light (fluorescence). This can be easily measured by com. fluorometers. Addnl. factors are the angle of radiation incidence, the placebo formula (vehicles), and other fluorescing products or ingredients. Light-protection factors measured by the biol. MED-based method and the described fluorescence method are comparable, though the values detd. by the latter using the described optical array tend to be higher. Water resistance and time-dependent changes of the protective effects of **sunscreens** are evaluated. The decrease of fluorescence caused by

desquamation and tape stripping is detd.

ST **sunscreen** light protective potential; fluorescence
sunscreen light protection

IT Fluorescence

(in light protective potential of **sunscreens** evaluation)

IT Sunburn and Suntan

(**sunscreens**, light protective potential of, evaluation of)

IT 605-65-2

RL: BIOL (Biological study)

(in light protective potential of **sunscreens** evaluation)

IT 830-09-1D, esters

RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(**sunscreens** contg., light protective potential of, evaluation
of)

L41 ANSWER 5 OF 32 CAPLUS COPYRIGHT 2003 ACS

AN 1997:376744 CAPLUS

DN 127:47140

TI **Sunscreens** prevent local and systemic immunosuppression of contact hypersensitivity in mice exposed to solar-simulated ultraviolet radiation

AU Roberts, Lee K.; Beasley, Donathan G.

CS Advanced Product Research, AR-3-59, Schering-Plough HealthCare Products, 3030 Jackson Avenue, Memphis, TN, 38151, USA

SO Journal of Photochemistry and Photobiology, B: Biology (1997), 39(2), 121-129

CODEN: JPPBEG; ISSN: 1011-1344

PB Elsevier

DT Journal

LA English

CC 8-7 (Radiation Biochemistry)

AB UV irradiation causes the immunosuppression of contact hypersensitivity (CH) responses in animals and humans. There are conflicting reports regarding the effectiveness of **sunscreens** in preventing UV-induced suppression of both local-type CH (induced by the application of the contact sensitizer directly to UV-exposed **skin**) and systemic-type CH (induced by the application of the contact sensitizer to an unirradiated **skin** site 3 days after UV exposure). The purposes of this study were as follows: 1. to derive solar simulator UV dose-response curves for the induction of local and systemic CH suppression in C3H mice; 2. to establish minimum immune suppression doses (MISDs) for local and systemic CH; 3. to determine the local and systemic immune protection capacity of two common **sunscreen** lotions with labeled sun protection factors (SPFs) of 4 and 8. Dose-response curves for the induction of local and systemic CH suppression were derived by exposing groups of mice to a range of full-spectrum UV doses (0.37-21.4 kJ m⁻²) on two consecutive days delivered from a filtered 1000 W xenon arc lamp solar simulator. The MISDs, defined as the lowest dose tested to cause approximately 50% suppression of the normal CH response, were obtained from the dose-response curves. Although the local and systemic immunosuppression dose-response curves were not statistically different, the MISD for local suppression of CH (1.35 kJ m⁻²) was about fivefold lower than that for systemic CH suppression (6.76 kJ m⁻²). The MISD was used as the endpoint to determine **sunscreen** immune protection levels. Both **sunscreens**, applied at 2 mg cm⁻², provided immune protection against the induction of local and systemic CH suppression in mice exposed to an effective UV dose of 1 MISD given through the **sunscreen**, i.e., 4 MISD to SPF 4 **sunscreen**-protected mice and 8 MISD to SPF 8 **sunscreen**-protected mice mounted CH responses that were significantly greater than those elicited in unprotected mice exposed to 1 MISD of solar-simulated UV radiation. The calculated immune protection factors for these **sunscreens** exceeded the level of protection predicted by their labeled SPFs, i.e. the local immune protection factor of both **sunscreens** was 15 and the systemic immune protection factors were 8 for the SPF 4 **sunscreen** and 15 for the SPF 8 **sunscreen**. Our data show that these two **sunscreens** provide levels of immune protection which exceed the levels predicted by their labeled SPFs in immunoprotection tests conducted in mice exposed to a relevant MISD of UV radiation from a source emitting a UV power spectrum similar to that of sunlight.

ST **sunscreen** immunosuppression solar UV

IT Dermatitis

(allergic, contact; **sunscreens** prevent local and systemic immunosuppression of contact hypersensitivity in mice exposed to solar-simulated UV radiation)

IT Solar UV radiation

Sunscreens

(**sunscreens** prevent local and systemic immunosuppression of contact hypersensitivity in mice exposed to solar-simulated UV

radiation)

IT Immunosuppression

(systemic; **sunscreens** prevent local and systemic immunosuppression of contact hypersensitivity in mice exposed to solar-simulated UV radiation)

IT 131-57-7, Oxybenzone 96436-87-2

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(**sunscreens** prevent local and systemic immunosuppression of contact hypersensitivity in mice exposed to solar-simulated UV radiation)

L41 ANSWER 4 OF 32 CAPLUS COPYRIGHT 2003 ACS
 AN 1998:608016 CAPLUS
 DN 129:347125
 TI Commercial **sunscreen** lotions prevent ultraviolet radiation-induced depletion of epidermal Langerhans cells in Skh-1 and C3H mice
 AU Beasley, D. G.; Montgomery, M. A.; Moloney, S. J.; Edmonds, S.; Roberts, L. K.
 CS Research and Development, Schering-Plough HealthCare Products, Memphis, TN, 38151, USA
 SO Photodermatology, Photoimmunology & Photomedicine (1998), 14(3/4), 90-99
 CODEN: PPPHEW; ISSN: 0905-4383
 PB Munksgaard International Publishers Ltd.
 DT Journal
 LA English
 CC 62-4 (Essential Oils and Cosmetics)
 Section cross-reference(s): 8
 AB There is much controversy regarding the ability of **sunscreens** to prevent UV (UV)-induced immune suppression. Epidermal Langerhans cells (LC) play a key antigen-presenting role in the afferent limb of the immune system's response to antigens introduced through the **skin**. It has been suggested that depletion of LC in UV-exposed **skin** is a crit. step toward the induction of immunosuppression by UV radiation. There are a no. of disparate reports with inconsistent results concerning the ability of **sunscreens** to prevent UV-induced depletion of LC. The purpose of this study was to systematically evaluate the ability of **sunscreens** to prevent UV-induced LC depletion in mice. Epidermal sheets obtained from **skin** biopsies taken from mice exposed to UV radiation from Kodacel-filtered FS20 sunlamps, which do not **emit** UV power at wavelengths <290 nm, were immunoperoxidase stained for LC using a rat monoclonal antibody against mouse Ia (major histocompatibility complex class II antigen). Time course and dose-response curves for LC depletion were generated for Skh-1 and C3H mice. Dose-response curves for acute UV exposure induced depletion of LC in Skh-1 and C3H mice were similar, but not identical. LC d. in the **skin** of Skh-1 mice that received chronic UV exposure (3 days/wk for 8 wk) was reduced by 62% after 2 wk of exposure, but returned to normal levels by 6 wk. Five com. **sunscreen** lotions with labeled sun protection factors (SPF) of 4, 8, 15, 30 and 45 were tested for their capacity to block UV-induced depletion of LC. LC were depleted .apprx.75% in the **skin** of unprotected or placebo lotion treated Skh-1 mice exposed to UV given on two consecutive days. Conversely, LC depletion was prevented in similarly UV exposed Skh-1 mice protected with a SPF 30 **sunscreen**. In C3H mice the levels of protection against LC depletion provided by the five **sunscreens** were proportional to the level of protection predicted by their labeled SPF. Comparisons of dose-response curves showed that significantly higher doses of UV were required for LC depletion and induction of **skin** edema than for the induction of local suppression of contact hypersensitivity. Thus, at UV doses where **sunscreens** provide complete protection against immunosuppression of contact hypersensitivity, prevention of LC depletion and **skin** edema would be expected.
 ST **sunscreen** epidermal Langerhans cell UV radiation
 IT **Sunscreens**
 (Coppertone; **sunscreen** lotions prevent UV radiation-induced depletion of epidermal Langerhans cells)
 IT **Skin**
 (Langerhans' cell; **sunscreen** lotions prevent UV radiation-induced depletion of epidermal Langerhans cells)
 IT Dermatitis
 (contact; **sunscreen** lotions prevent UV radiation-induced depletion of epidermal Langerhans cells)
 IT **Skin**, disease

Skin, disease

(edema; **sunscreen** lotions prevent UV radiation-induced depletion of epidermal Langerhans cells)

IT **Skin**
(epidermis; **sunscreen** lotions prevent UV radiation-induced depletion of epidermal Langerhans cells)

IT Immunosuppression

Skin

UV radiation

(**sunscreen** lotions prevent UV radiation-induced depletion of epidermal Langerhans cells)

IT 118-56-9, Homosalate 118-60-5, Octyl salicylate 131-57-7, Oxybenzone 5466-77-3, Octyl p-methoxycinnamate 6197-30-4, Octocrylene
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(**sunscreen** lotions prevent UV radiation-induced depletion of epidermal Langerhans cells)

RE.CNT 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD

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L41 ANSWER 3 OF 32 CAPLUS COPYRIGHT 2003 ACS

AN 2000:10521 CAPLUS

DN 132:69093

TI **Skin** protectant comprising 5-substituted and 5,5-disubstituted
3,4-dihydroxy-2(5H)-furanones

IN Ziemniak, John A.; Hopper, Allen T.; Pugliese, Peter T.

PA Oxis International, Inc., USA

SO U.S., 6 pp.

CODEN: USXXAM

DT Patent

LA English

IC ICM A61K007-42

ICS A61K007-00; A61K031-34

NCL 424059000

CC 62-4 (Essential Oils and Cosmetics)

Section cross-reference(s): 1, 63

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 6010685	A	20000104	US 1999-264659	19990308
PRAI	US 1999-264659		19990308		

OS MARPAT 132:69093

AB Formulations of racemic or optically active 3,4-dihydroxy-5-aryl-2(5H)-furanones are provided for topical administration to the **skin** for the inhibition and prevention of sunburn cell formation resulting from exposure to UV radiation and other sources of damage, and for the treatment of **skin** aging. The compds. may be formulated in combination with a **sunscreen**, and may be applied before, during, and/or after exposure. Male and female hairless mice of at least 8 wk old were used in the study. Male and female hairless mice of at least 8 wk old were used in the study. The mice were conditioned in the lab. for 1-2 wk, and subjected to a 24-h day/night cycle. 5-[(1,1'-Biphenyl)-4-yl]-3,4-dihydroxy-2(5H)-furanone (BPHTA) was prepd. as a formulation in a mixt. of 50% oleic acid and 50% SD39 (denatured ethanol). Formulations contg. 1% BPHTA and 2.5% BPHTA were prepd. Two mice were used for each test compn.; two mice untreated before UV irradiation served as controls. UV lamps emitting UVB, were used to supply the UV energy. The mice were exposed to UVB for 1.75 min for a dose of 70 mJ/cm², and then sacrificed 24 h later by cervical dislocation. The **skin** was removed and processed for histol. staining (hematoxylin and eosin). The percent redn. was 31 and 69, resp., for the 2 BHPA formulations.

ST furanone hydroxy **skin** protectant UV; **sunscreen**

IT **Skin**, disease

(aging; **skin** protectant compns. contg. dihydroxyfuranones)

IT Drug delivery systems

Photoprotectants

Sunscreens

(**skin** protectant compns. contg. dihydroxyfuranones)

IT Drug delivery systems

(topical; **skin** protectant compns. contg. dihydroxyfuranones)

IT 79821-07-1 139572-59-1 203863-19-8 203863-20-1 203863-22-3

210557-46-3 253325-76-7 253325-77-8

RL: BUU (Biological use, unclassified); THU (Therapeutic use); BIOL
(Biological study); USES (Uses)

(**skin** protectant compns. contg. dihydroxyfuranones)

RE.CNT 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

(1) Wenk; US 4426380 1984 CAPLUS

L41 ANSWER 2 OF 32 CAPLUS COPYRIGHT 2003 ACS
 AN 2001:213008 CAPLUS
 DN 136:50588
 TI Photoacoustic trace gas detection of ethene released by UV-induced lipid peroxidation in humans
 AU Cristescu, Simona M.; Berkelmans, Rik; te Lintel Hekkert, Sacco; Timmerman, Brenda H.; Parker, David H.; Harren, Frans J. M.
 CS Dept. of Molecular and Laser Physics, Univ. of Nijmegen, Nijmegen, 6500 GL, Neth.
 SO Proceedings of SPIE-The International Society for Optical Engineering (2000), 4162(Controlling Tissue Optical Properties), 101-107
 CODEN: PSISDG; ISSN: 0277-786X
 PB SPIE-The International Society for Optical Engineering
 DT Journal
 LA English
 CC 9-16 (Biochemical Methods)
 Section cross-reference(s): 8
 AB A sensitive CO₂ laser-based photoacoustic (PA) detector has been used to perform non-invasive and online measurements of ethene (C₂H₄) prodn. from exhaled air and directly **emitted** from the **skin**. Ethene was used as indicator for free-radicals induced lipid peroxidn. in the **skin** of human subjects exposed to UV radiation from a solarium. Ethene from the exhaled air was analyzed for a group of 21 male subjects at rest. During 15 min of UV exposure, the av. ethene emission was 17.2 pmol/kg/min (SD 7.3), while the pre-UV exposure levels were 1.4 pmol/kg/min (SD 0.38). Different types of sun protection creams were tested by means of ethene release in exhaled air. The influence of UV radiation intensity and of exposure time (10 and 15 min, resp.) on the ethene emission from the **skin** has been studied for a second group of 12 subjects. Comparison between measurements of exhaled air and directly on the **skin** is presented.
 ST photoacoustic gas detection ethene UV lipid peroxidn
 IT Lasers
 (carbon dioxide; photoacoustic trace gas detection of ethene released by UV-induced lipid peroxidn. in humans)
 IT Respiratory air
 (exhaled; photoacoustic trace gas detection of ethene released by UV-induced lipid peroxidn. in humans)
 IT Peroxidation
 (lipid; photoacoustic trace gas detection of ethene released by UV-induced lipid peroxidn. in humans)
 IT Lipids, biological studies
 RL: BSU (Biological study, unclassified); BIOL (Biological study)
 (peroxidn.; photoacoustic trace gas detection of ethene released by UV-induced lipid peroxidn. in humans)
 IT Acoustic devices
 (photoacoustic devices; photoacoustic trace gas detection of ethene released by UV-induced lipid peroxidn. in humans)
 IT Human
 Skin
 Sunscreens
 Time
 UV radiation
 (photoacoustic trace gas detection of ethene released by UV-induced lipid peroxidn. in humans)
 IT 74-85-1, Ethene, analysis
 RL: ANT (Analyte); ANST (Analytical study)
 (photoacoustic trace gas detection of ethene released by UV-induced lipid peroxidn. in humans)
 RE.CNT 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD
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L41 ANSWER 1 OF 32 CAPLUS COPYRIGHT 2003 ACS

AN 2002:10251 CAPLUS

DN 136:74320

TI Cosmetic and pharmaceutical compositions containing green-light
emitting materials

IN Rein, Glen; George, Liliana; Cioca, Gheorge; Comorosan, Sorin

PA E-L Management Corp., USA

SO PCT Int. Appl., 21 pp.

CODEN: PIXXD2

DT Patent

LA English

IC ICM A61K007-48

ICS A61K033-04; A61K033-06; A61K033-22; A61K033-30

CC 62-4 (Essential Oils and Cosmetics)

Section cross-reference(s): 63

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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PI	WO 2002000190	A1	20020103	WO 2001-US11612	20010410
	W: AU, CA, JP				
	RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR				
	EP 1296639	A1	20030402	EP 2001-923244	20010410
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY, TR				
PRAI	US 2000-604536	A	20000627		
	WO 2001-US11612	W	20010410		
AB	The present invention relates to cosmetic and pharmaceutical compns. and methods for use of green-light emitting materials. Based on the obsd. effect of green light on oxygen radical concn. in treated skin cells vs. untreated controls, the green-light emitting material can be used as an antioxidant to protect the skin cells against damage caused by oxygen free radicals, as well as protecting the skin against the damaging effects of UV radiation. Examples of green light emitting materials include green phosphorescent or fluorescent materials, such as pigments or minerals. The green light had a protective effect against UV damage. However, the magnitude of the protective effect was dependent on the dose, with green light being less protective against higher doses of UVB.				
ST	green light emitting pigment pharmaceutical cosmetic; mineral green light antioxidant				
IT	Antioxidants Cosmetics Drug delivery systems Fluorescent substances Phosphorescent substances Pigments, nonbiological Sunscreens (cosmetic and pharmaceutical compns. contg. green-light emitting materials)				
IT	Minerals, biological studies Oxides (inorganic), biological studies Rare earth metals, biological studies RL: COS (Cosmetic use); THU (Therapeutic use); BIOL (Biological study); USES (Uses) (cosmetic and pharmaceutical compns. contg. green-light emitting materials)				
IT	Light Pigments, nonbiological (green; cosmetic and pharmaceutical compns. contg. green-light emitting materials)				
IT	Pigments, nonbiological (phosphorescent; cosmetic and pharmaceutical compns. contg. green-light				

emitting materials)

IT Metals, biological studies
 RL: COS (Cosmetic use); THU (Therapeutic use); BIOL (Biological study);
 USES (Uses)
 (plated with copper or rare earth metal; cosmetic and pharmaceutical
 compns. contg. green-light **emitting materials)**

IT 1314-98-3, Zinc sulfide, biological studies
 RL: COS (Cosmetic use); THU (Therapeutic use); BIOL (Biological study);
 USES (Uses)
 (copper- or manganese-doped; cosmetic and pharmaceutical compns. contg.
 green-light **emitting materials)**

IT 37341-47-2, Zinc borosilicate
 RL: COS (Cosmetic use); THU (Therapeutic use); BIOL (Biological study);
 USES (Uses)
 (copper- or rare earth metal-doped; cosmetic and pharmaceutical compns.
 contg. green-light **emitting materials)**

IT 7782-44-7, Oxygen, biological studies
 RL: BSU (Biological study, unclassified); BIOL (Biological study)
 (cosmetic and pharmaceutical compns. contg. green-light
emitting materials)

IT 7440-46-2D, Cesium, salts 13397-26-7, Calcite, biological studies
 42617-47-0, Strontium aluminate
 RL: COS (Cosmetic use); THU (Therapeutic use); BIOL (Biological study);
 USES (Uses)
 (cosmetic and pharmaceutical compns. contg. green-light
emitting materials)

RE.CNT 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

- (1) Bruckner; US 5562763 A 1996 CAPLUS
- (2) Kosei Co; JP 10-330209 A CAPLUS 1998:799831 1998 CAPLUS
- (3) Lumbroso; FR 1528072 A 1968 CAPLUS
- (4) L'Oreal; FR 2772770 A 1999 CAPLUS
- (5) Patent-Treuhand-Gesellschaft Fur Elektrische Gluhlampen; GB 2209624 A 1989
- (6) Pennzoil Products Company; WO 9838981 A 1998 CAPLUS
- (7) Pyramid Productions; WO 0056274 A 2000 CAPLUS
- (8) Sri International; WO 9929801 A 1999 CAPLUS